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**Issue date: 15Jun2001**

CASE NO. 1999-BLA-544

In the Matter of

GRACE POPICH Survivor of STEVE J. POPICH,
Claimant

v.

CONSOLIDATION COAL COMPANY,
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest

Appearances:

Charles Evans, Esq.
For the Claimant

George Stipanovich, Esq.
For the Employer/Carrier

Before: RICHARD A. MORGAN
Administrative Law Judge

## DECISION AND ORDER ON REMAND DENYING BENEFITS

This proceeding arises from a survivor's claim for benefits, under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq.* ("Act"), filed on December 11, 1997. The Act and implementing regulations, 20 C.F.R. parts 410, 718, and 727 (Regulations), provide compensation and other benefits to:

1. Living coal miners who are totally disabled due to coal workers' pneumoconiosis ("CWP") and their dependents;

2. Surviving dependents of coal miners whose death was due to pneumoconiosis; and,

3. Surviving dependents of coal miners who were totally disabled due to pneumoconiosis at the time of their death.

The revised Act and Regulations define pneumoconiosis as encompassing both "clinical" and "legal" pneumoconiosis. Clinical pneumoconiosis includes those coal dust related diseases recognized by the medical community, and legal pneumoconiosis refers to any chronic dust disease of the lungs and its sequelae, including but not limited to any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment. A dust disease of the lungs is considered to arise out of coal mine employment if the disease is significantly related to or substantially aggravated by dust exposure in coal mine employment. Pneumoconiosis is considered to be a latent and progressive disease that may first become detectable only after cessation of coal mine dust exposure. 20 C.F.R. § 718.201(a) et. seq.

## PROCEDURAL HISTORY

The claimant is the widow of Steve Popich. She filed the instant survivor's claim for federal black lung benefits on December 11, 1997. (Director's Exhibit "DX" 1). On July 26, 1999, I held a formal hearing in Pittsburgh, Pennsylvania, in which the parties had an opportunity to present evidence and testimony. I issued a formal Decision and Order Denying Benefits on August 13, 1999. Subsequently, the claimant, through counsel, filed a timely appeal to the Benefits Review Board. Employer filed a cross-appeal. The Board, in a Decision and Order dated December 28, 2000, affirmed my decision in part, vacated it in part, and remanded the case back to me for further consideration.

The claimant challenged my reliance on Drs. Kleinerman and Mendelow to support my finding that death was not causally related to pneumoconiosis because I had found the presence of pneumoconiosis even though neither Drs. Kleinerman nor Mendelow did so. The employer's cross-appeal challenged my finding of coal workers' pneumoconiosis pursuant to 718.202(a)(2).

The Board noted that no party challenged my finding of no pneumoconiosis pursuant to Sections 718.202(a)(1), (a)(3), and (a)(4) and therefore affirmed those findings. However, the Board instructed that I reconsider my findings of coal workers' pneumoconiosis pursuant to 718.202(a)(2). Specifically, I am to discuss the evidence pertaining to anthracosis and whether it qualifies as a diagnosis of coal workers' pneumoconiosis. I am also to discuss in greater detail the contrary opinions of Drs. Kleinerman and Mendelow and the weight I assign to their opinions. I am to discuss all of the relevant evidence pertaining to 718.202(a)(2) and provide specific bases for my conclusions. If, upon reconsideration, I again find that the claimant established disease presence, I am to reweigh all of the evidence at 718.202(a)(1)-(4) together in accordance with *Penn Allegheny Coal Co. v. Williams,* 114 F.3d 22, 21 B.L.R. 2-104 (3d Cir. 1997). Finally, if I again find that the miner had pneumoconiosis at the time of death, I am to reconsider the evidence at 718.205(c)(2). Moreover, if I

do find the presence of coal workers' pneumoconiosis pursuant to 718.202(a), I am to resolve that finding against the opinions of Drs. Kleinerman and Mendelow, both of whom found that coal workers' pneumoconiosis played no part in the miner's death.

It should be noted that the Board affirmed all of my other findings of fact, including the miner's status as a coal miner; the dependency status and eligibility of his widow, Grace; the coal company's status as responsible operator; and the finding that the miner worked for 25 years in the mines.

## ISSUES

I. Whether the decedent miner had pneumoconiosis as defined by the Act and the Regulations?

II. Whether the decedent miner's pneumoconiosis arose out of his coal mine employment?

III. Whether the decedent miner's death was due to pneumoconiosis?

## FINDINGS OF FACT

To briefly recap my prior findings regarding the non-medical issues, it should be noted that the miner died on March 20, 1997 at the age of 80. He last worked as a miner on September 6, 1980 and retired upon closure of the mine in which he was employed, the responsible operator in this matter. The miner was credited with twenty-five years of coal mine employment. He had filed a living miner's federal black lung claim on October 2, 1980, although the same was denied by Administrative Law Judge Daniel Sarno. Judge Sarno did not make a finding of pneumoconiosis and therefore, denied benefits. The denial was affirmed on appeal via Decision and Order of the Benefits Review Board on May 31, 1990.

### *Review of Pathologic Evidence*

The autopsy was performed by Dr. Hamada Mahmoud, and the autopsy materials and protocol were reviewed by Dr. A. Shakir. (DX 13). Both of these pathologists work out of the Allegheny County Coroner's Office, headed by Dr. Cyril Wecht. The protocol contains a gross and a microscopic description. I would note that this was a full autopsy with no apparent limitations to any particular area of the body. On gross examination, it should be noted that Dr. Mahmoud found mild dilatation of the right ventricle although the right and left ventricles were not hypertrophied. The coronary arteries showed moderate or severe atherosclerosis and stenosis. The gross description of the lungs revealed multiple emphysematous bullae and scattered fibrotic nodules measuring up to 0.7 centimeters, mainly in the upper lobes. He noted moderately congested and edematous lung tissue. He noted that the hilar and mediastinal lymph nodes are enlarged and show marked anthracosis. On microscopic examination, particularly of the lungs, Dr. Mahmoud found panlobular emphysema, interstitial fibrosis in some areas and reactive hyperplasia of the hilar and mediastinal lymph nodes.

The final anatomic diagnoses set forth in the March 20, 1997 protocol included arteriosclerotic cardiovascular disease, which encompassed atherosclerosis and stenosis of the left, circumflex and right coronary arteries and well as of the aorta. He also had arteriolonephrosclerosis. Cor pulmonale was not listed as a diagnosis. The prosector also found that the miner had chronic obstructive lung disease *clinically*, and specified emphysema of the lungs and marked anthracosis. Other diagnoses included cirrhosis of the liver, hemorrhagic gastritis, hemorrhagic cystitis and recent history of right hip fracture. The opinion of the prosector was that the miner died as a result of arteriosclerotic cardiovascular disease and cirrhosis of the liver as a contributory cause.

The death certificate was signed by Dr. Kenneth Suchan, deputy coroner. (DX 11). It is noted that Dr. Cyril Wecht completed the cause of death, although Dr. Wecht's signature does not appear on the certificate. The cause of death on the certificate mirrors that which was noted in the autopsy, namely, arteriosclerotic cardiovascular disease as the immediate cause of death, and cirrhosis of the liver as a significant condition contributing to death.

On behalf of the U.S. Department of Labor, Dr. Joshua Perper reviewed the autopsy slides and many medical records. He issued a report dated December 1, 1998. Although Dr. Perper's curriculum vitae is not a part of the record, I take judicial notice that Dr. Perper is Board certified in the field of pathology. In his review of the records he noted the miner's previous treatment and hospitalization for exacerbation of COPD. He also noted a smoking history in the range of 15 pack years to approximately 120 pack years. Dr. Perper reviewed twelve slides, ten of which were of the lungs and contained sixteen lung sections. His diagnoses were: simple coal workers' pneumoconiosis with severe interstitial fibrosis and deposition of anthracosilicotic deposits and a few micronodules; marked pneumoconiotic involvement of hilar lymph nodes by pneumoconiotic micronodules and macronodules with silica crystals; moderate to marked centrilobular emphysema; severe pan-lobular emphysema with bullae; sclerosis of intra-pulmonary blood vessels; and small foci of organizing pneumonia. In his microscopic review, Dr. Perper described a small fibro-anthracotic micronodule measuring 1 millimeter in the pleura and another 3 millimeter micronodules in the right lung. He found a macronodule measuring up to 7 millimeters in the right hilar lymph node. He also found a few micronodules, which he presumed were in the left lung, measuring up to 3-4 millimeters.

Dr. Perper concluded that the miner had evidence of severe chronic obstructive interstitial lung disease as evidenced by all of the medical documentation. He did diagnose coal dust related lung disease based on history, symptoms, clinical manifestations of respiratory impairment, and pathological findings. It was Dr. Perper's opinion that the prosector likely missed the diagnosis of coal workers' pneumoconiosis because he was an in-training pathology fellow, and that the staff pathologist did not thoroughly review the microscopic findings. He added that the autopsy report failed to mention that polarized light was used to examine for silica crystals. Dr. Perper then went on to cite numerous studies regarding the relationship between coal dust and diffuse interstitial fibrosis as well as coal dust and centrilobular emphysema. It was Dr. Perper's opinion that the miner's "significant" coal workers' pneumoconiosis with associated interstitial fibrosis and centrilobular emphysema was a substantial

factor in the miner's pulmonary disability and death. He said it was medically impossible to separate out the effects of smoking from the effects of coal dust exposure.

The autopsy slides and medical records were reviewed by Dr. Harvey Mendelow at the request of the Employer. Dr. Mendelow is Board-certified in Anatomic and Clinical Pathology. Dr. Mendelow issued a report dated June 25, 1999. Dr. Mendelow reported that he received 26 tissue slides, including 8 slides of 12 samples containing lung tissue. The records that he reviewed included hospital records and various clinical studies such as chest x-ray and CT scan reports as well as pulmonary function and arterial blood gas studies. He demonstrated an awareness of the miner's work history and job duties as well as his medical history. Through review of records, Dr. Mendelow concluded that the decedent miner had smoked somewhere between 60 and 120 pack years. He noted that the majority of the chest x-ray readings were not diagnostic of coal workers' pneumoconiosis. He added that the pulmonary function studies indicated a worsening which was attributed to emphysema from cigarette smoking. He observed that up until 1987 the miner's arterial blood gas studies revealed no evidence of hypoxemia. It wasn't until after 1992 that the miner began to be hospitalized for increased respiratory symptoms.

Dr. Mendelow described his findings upon review of the lung slides. He found severe panlobular and irregular emphysema in all sections, including "smoke macrophages," characteristic of heavy smoking. He said that the most severe emphysematous areas also contain interstitial fibrosis and scar formation. He further found evidence of acute passive congestion due to terminal congestive heart failure and organizing and acute pneumonia. Other findings made by Dr. Mendelow in the lungs included scant anthracotic pigment, found in macrophages and occasionally in some of the interstitial fibrotic scars. He said there were no macules or micronodules of even the most minimal degree of simple coal workers' pneumoconiosis. Although he found a few fibrotic granulomas with peripheral trapped anthracotic pigment, he noted that they were simple silicotic granulomas that were of little functional significance.

Dr. Mendelow concluded that the cause of the miner's death was cardio-respiratory failure from end-stage chronic obstructive pulmonary disease complicated by renal failure, congestive heart failure, bronchopneumonia and the metabolic complications of alcoholic cirrhosis. As for the primary etiology of the COPD, Dr. Mendelow opined that it was due to the decedent's heavy smoking history of up to 120 pack years. He found no substantial evidence of even minimal simple pneumoconiosis and thus eliminated this disease as a factor in the miner's death. He said that the miner would have died at the same time and in the same manner had he never mined coal.

Dr. Jerome Kleinerman, also Board-certified in Anatomic and Clinical Pathology, reviewed extensive medical records, the autopsy slides, fifteen chest x-rays ranging between 1980-1994, and twelve portable films ranging between January 5 and March 20, 1997. He subsequently issued a report dated June 25, 1999. In great detail, he summarized the medical records he reviewed and succinctly reported his understanding of the miner's past medical and smoking history. In particular,

Dr. Kleinerman noted that the miner had a smoking history of anywhere from 15-120 pack years, and was still smoking several months before his death. The miner had been treated for COPD in his lifetime.

Dr. Kleinerman did not diagnose pneumoconiosis upon his review of all of the chest x-rays. He did note evidence of emphysema on some of them as well as infiltrates of the right upper and lower lung fields. He observed a density in the right perihilar region in a film dated March 6, 1997 which he said had not been present earlier. However, he acknowledged that many of the portable films were of poor quality or unreadable.

Dr. Kleinerman reviewed 26 autopsy slides, including 8 slides of 15 different sections of lung tissue from all parts of the lungs. Dr. Kleinerman found intraalveolar hemorrhage with organizing pneumonitis and interstitial fibrosis. He diagnosed chronic bronchitis. He found minimal black granular pigment in the subpleural lung tissue, in the bronchial lumen and in macrophages. On several slides he found extensive panacinar emphysema. He did not diagnose simple or complicated coal workers' pneumoconiosis. Upon review of the heart sections, he found mild or marked atherosclerosis.

It was Dr. Kleinerman's opinion that the miner's death was due to cardiac arrest following cardiac arrhythmias including ventricular tachycardia and ventricular fibrillation. It was his opinion that the miner's respiratory impairment was from the diffuse interstitial fibrosis, panacinar emphysema and chronic bronchitis. He emphasized that there was no evidence of pneumoconiosis and that cigarette smoking caused the obstruction, not coal mine dust. He referred to a Surgeon General's report indicating that 20 years of cigarette smoking is the major cause of chronic obstructive lung disease, centriacinar and panacinar emphysema as well as coronary artery atherosclerosis. Dr. Kleinerman noted that the miner's pulmonary function tests were normal up to five years after he left the mines and that the arterial blood gas studies were normal 16 years after he left the mines. He alluded to "scholarly medical literature" that simple CWP does not progress after a miner leaves the coal mines and concluded that the miner's lung dysfunction did not result from coal mine dust exposure. He said that with reasonable medical certainty, the miner's pulmonary dysfunction was due to his prolonged heavy cigarette smoking history.

Dr. Kleinerman issued a report dated June 26, 1999 (EX B) in which he critiqued the report of Dr. Joshua Perper. Basically, Dr. Kleinerman refuted numerous studies cited by Dr. Perper in support of several positions he advanced in his report addressed to the United States Department of Labor. For example, Dr. Kleinerman disagreed with Dr. Perper's position that the miner's coal dust exposure caused his centriacinar emphysema. He said that the studies relied upon by Dr. Perper in support of this position were flawed for various reasons, such as the fact that cigarette smoking was not properly accounted for, or because the studies focused on focal but not centriacinar emphysema. Dr. Kleinerman reviewed and critiqued these studies with painstaking detail. Dr. Kleinerman also disagreed with Dr. Perper's position that diffuse interstitial fibrosis may be caused by coal mine dust. Again, Dr. Kleinerman carefully deconstructed the medical literature relied upon by Dr. Perper, pointed to various biases in these studies, and concluded that there was no evidence that diffuse interstitial fibrosis occurs more frequently among coal miners than in the general population. Finally, he emphatically disagreed

with Dr. Perper's diagnosis of simple coal workers' pneumoconiosis, arguing that it was not present on the lung tissue slides, and Dr. Perper was incorrect in believing that the prosector overlooked this diagnosis. He said that the signature of a staff pathologist indicated that the protocol was approved and reviewed. Dr. Kleinerman concluded that Dr. Perper did not provide scientifically valid support for his medical conclusions. He said that since CWP was not present at autopsy, it could not have been a substantial cause of the miner's death.

*III. Conclusions of Law*

The Black Lung Statute, found at 30 U.S.C. § 902(b) and its implementing regulations, found at 20 C.F.R. § 718.201, define pneumoconiosis as a "a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment."[1] The definition is not confined to "coal workers' pneumoconiosis," but also includes other diseases arising out of coal mine employment, such as anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis, or silicotuberculosis. 20 C.F.R. § 718.201. The term "arising out of coal mine employment" includes "any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."

New black lung regulations were enacted recently by the Department of Labor, effective January 19, 2001. However, not all sections were retroactively applicable to cases pending at the time they were enacted. The United States District Court for the District of Columbia, on February 9, 2001, issued a Preliminary Injunction Order stating that all pending black lung cases shall be stayed unless the adjudicator determines that the specific regulations at issue will not affect the outcome of the case.

On March 9, 2001, I issued an Order directing the parties to brief their respective positions as to whether or not the new regulations would affect the outcome of the case and if so, in what way. The Director replied that the new regulations will not affect the outcome of the case because they do not materially change the standards for determining death due to pneumoconiosis in the Third Circuit, in whose jurisdiction this case arises. Also, there were no treating physicians' opinions in this case. She also argued that the definition of pneumoconiosis, now codified by the new regulations, which makes a distinction between legal and clinical pneumoconiosis, has already been recognized by the Third Circuit. She made a similar argument concerning the codification of the principle that pneumoconiosis is a latent and progressive disease in that the Third Circuit has also adjudicated this issue. Both the Claimant and the Employer informed the undersigned that they agreed with the Director's position that the new regulations would not affect the outcome of the case.

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[1] Pneumoconiosis is a progressive and irreversible disease; once present, it does not go away. *Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 151 (1987); *Lisa Lee Mines v. Director*, 86 F.3d 1358, 1364 (4th Cir. 1996)(*en banc*); *LaBelle Processing Co. v. Swarrow*, 72 F.3d 308, 314-15 (3rd Cir. 1995).

After considering the Director's argument and careful review of the regulations, it is also my conclusion that the aforementioned regulations would not have made a difference on the merits. I agree with the position taken by the Director in her brief as it pertains to the instant case. The regulations in question did not affect the weight I gave to any of the evidence, nor did it alter my decision in any way.

The new regulations set forth a broader definition of what constitutes pneumoconiosis. A distinction first must be made between "legal" and "clinical" pneumoconiosis. In particular, one may have "legal" pneumoconiosis but not "clinical" or medical pneumoconiosis. Legal pneumoconiosis is that which arises out of coal mine employment and can include chronic obstructive pulmonary disease. The key issue is whether a particular lung or respiratory disease actually arose out of coal mine employment. For example, one may have acquired chronic obstructive pulmonary disease as a result of exposure to coal dust, which would constitute legal pneumoconiosis. However a coal miner may also have contracted the disease elsewhere, and if he has not established by the preponderance of the evidence that his COPD was caused or contributed to by coal dust, then he cannot establish legal pneumoconiosis.

A survivor's first burden is to establish the existence of pneumoconiosis. The regulations provide the four means of establishing the existence of pneumoconiosis by: (1) a chest x-ray meeting the criteria set forth in 20 C.F.R. § 718.202(a); (2) a biopsy or autopsy conducted and reported in compliance with 20 C.F.R. § 718.106; (3) application of the irrebuttable presumption for "complicated pneumoconiosis" found in 20 C.F.R. § 718.304; or (4) a determination of the existence of pneumoconiosis made by a physician exercising sound judgment, based upon certain clinical data and medical and work histories, and supported by a reasoned medical opinion. 20 C.F.R. § 718.202(a).

Section 718.202 provides that the existence of pneumoconiosis can be established by x-ray, autopsy, or biopsy; by the presumptions contained in Sections 718.304, 718.305, 718.306; or by the report of a physician exercising sound medical judgment stating that the claimant suffers from pneumoconiosis. Pneumoconiosis must be proved by a preponderance of the evidence, and the fact-finder must weigh all types of relevant evidence together to determine whether the miner suffers from this disease. *See Penn Allegheny Coal Co. v. Director, OWCP,* 114 F.3d 22 (3d Cir. 1997).

In this particular case, I have already indicated that the claimant has failed to establish that her late husband had legal or clinical pneumoconiosis through x-ray evidence, biopsy evidence, or reasoned medical opinion evidence. I had previously determined that the autopsy evidence had revealed the presence of pneumoconiosis, in large part because of the prosector's finding of marked anthracosis. Upon re-review of the post-mortem evidence, it is now my opinion that the miner has failed to establish disease presence pursuant to 718.202(a)(2).

Although anthracosis is specifically enumerated in the regulations as falling within the legal definition of coal workers' pneumoconiosis, there is no evidence that this finding made upon autopsy was actually a byproduct of the miner's coal mine employment. I hold this opinion for several reasons. First, a distinction should be made between anthracosis and anthracotic pigmentation. Anthracosis meets the legal definition of pneumoconiosis, but anthracotic pigmentation does not. See *Bueno v.*

*Director, O.W.C.P.,* 7 B.L.R. 1-337 (1984)*; Dagan v. Black Diamond Coal Mining Co.,* 994 F2d 1536 (11th Cir. 1993). When anthracosis is diagnosed in the lymph nodes, the administrative law judge must resolve the question of whether this constitutes pneumoconiosis. See *Mangus v. Director, O.W.C.P.,* 882 F.2d 1527, 13 B.L.R. 2-9 (10th Cir. 1989) (anthracosilicosis found in the lymph nodes was statutory pneumoconiosis based upon the detailed deposition of the surgeon, the detailed pathology report and an additional letter of explanation). See also *Dobrosky v. Director, O.W.C.P.,* 4 B.L.R. 1-680 (1982). It should be noted that the Third Circuit has not spoken on the issue of the definition of anthracosis.

In this particular case, anthracosis was discovered in the hilar lymph nodes, but in the protocol, the prosector made no mention of anthracosis in the lung tissue itself. As for his microscopic description, the pathologist did not make any mention of anthracosis or classic coal workers' pneumoconiosis. He did not even describe nodules microscopically, although he did indicate interstitial fibrosis and panlobular emphysema, but there is no convincing evidence that he was referring to pneumoconiosis from either a medical or legal standpoint. Both Dr. Mendelow and Dr. Kleinerman did not note anthracosis and only found scant black granular pigmentation. Neither of these physicians found any nodular changes consistent with pneumoconiosis and in fact, both doctors specifically stated that there was no medical coal workers' pneumoconiosis. Even Dr. Perper only found slight anthracotic pigmentation, although he did find anthracotic micronodules.

I do not believe that the prosector's diagnosis of marked anthracosis in the hilar lymph nodes is equivalent to a legal or clinical definition of pneumoconiosis in part because there is no other support in the protocol of legal or clinical pneumoconiosis, even upon microscopic review. Also, the two pathologists to whom I give the most weight, Drs. Kleinerman and Mendelow, did not make this finding even though they reviewed slides of the lymph nodes.

Moreover, for anthracosis to be equivalent to legal pneumoconiosis, certain criteria must be met. I had previously referenced the case of *Brooks v. W.P. Coal Co.,* 110 F.3d 59, 1997 U.S. App. LEXIS 647 (4th Cir. 1997) (unpublished) as standing for the proposition that anthracosis may only be considered as legal pneumoconiosis if it results in respiratory or pulmonary impairment and is significantly related to or substantially aggravated by coal dust exposure. The existence of the disease on autopsy is not necessarily dispositive of its causal nexus to coal dust exposure, particularly, as has been pointed out, since it was found only in the hilar lymph nodes. Other than Dr. Perper, no other pathologist, including the prosector, related the anthracosis to coal dust exposure. Moreover, neither Dr. Kleinerman nor Dr. Mendelow even found the existence of a coal dust related disease. The prosector did not comment as to whether or not the anthracosis found was related to coal dust exposure, but, without supporting evidence, I cannot find that it meets the legal definition. This is particularly true because there is no evidence of any significant respiratory impairment due to anthracosis or any coal dust related disease. The most recent objective pulmonary function study did not conform to the regulations as the tracings were not available. Moreover, the majority of the most recent arterial blood gas studies, as noted in my original decision, had failed to yield results which would have qualified him for disability. Any respiratory impairment to the miner's smoking history, as he had a longstanding smoking history which is undisputed by the evidence of record. In that there is no

evidence of significant respiratory impairment related to coal dust exposure, I cannot find that the anthracosis meets the definition of pneumoconiosis.

I give great weight to the opinions of Drs. Kleinerman and Mendelow on both the issues of disease presence and cause of death. Both of these physicians are eminently qualified as experts in their field. Moreover, the opinions of Drs. Kleinerman and Mendelow were mutually consistent. Although Dr. Perper is also well-qualified, I place less weight on his overall opinion for several reasons. First, he found centrilobular emphysema, which no other physician found. He also found nodules relating to coal dust exposure, which again, no other physician found. I also disagree with Dr. Perper's theory that the prosector missed the diagnosis of pneumoconiosis due to his status as a fellow rather than as a more experienced staff pathologist. Dr. Perper surmised that the staff pathologist did not thoroughly review the findings of Dr. Mahmoud. This is nothing more than speculative on Dr. Perper's part and I find nothing to support his presumption. Although his credentials and curriculum vitae were not made a part of the record, there is no evidence that Dr. Mamoud was not qualified to perform the autopsy. I previously find, and I continue to do so, that his findings were reviewed by Dr. Shakir. Therefore, I give great weight to the prosector, who did not implicate any coal dust related disease in making his conclusions as to cause of death. The prosector's findings on microscopic examination were highly consistent with the findings made by Drs. Kleinerman and Mendelow.

It is acknowledged that the miner also had emphysema. Under the new regulations, and codified by case law prior to the enactment of the regulations, emphysema, or chronic obstructive pulmonary disease, can be considered legal pneumoconiosis, again if there is a coal dust origin or exacerbation of the disease. The pathologists disagree as to the type of emphysema that the miner had. The prosector and Dr. Mendelow found panlobular emphysema, while Dr. Kleinerman found panacinar emphysema. Dr. Perper also found panlobular emphysema but also described emphysema of the centrilobular type.

I continue to find that the miner's emphysema and chronic obstructive pulmonary disease was not equivalent to coal workers' pneumoconiosis. As I explained previously, the miner had an extensive smoking history, which I found to have substantially accounted for his chronic obstructive pulmonary disease.

Therefore, I find that the miner has failed to establish coal workers' pneumoconiosis via 718.202(a)(2). In my prior decision, I had found that disease presence was not established via 718.202(a)(1), (a)(3), or (a)(4), and these findings were affirmed by the Board. In that I now have found that pneumoconiosis has not been proven via autopsy evidence per 718.202(a)(2), there is no need to further weigh all of the evidence together to determine if pneumoconiosis is present.

Even if I had found the presence of pneumoconiosis to exist again, that would not have changed my overall decision to deny benefits. In order to be awarded benefits, a survivor must establish that pneumoconiosis caused, substantially contributed to, or hastened the miner's death. See *Lukosevicz v. Director, OWCP*, 888 F.2d 1001 (3d Cir. 1989). This standard has not changed since the implementation of the new regulations. For decedent miner's widow to have been awarded benefits, I would have had to have credited Dr. Perper with the most weight as he was the only physician to have

found pneumoconiosis to have been responsible for the miner's death. I also would have had to find that Dr. Perper's opinion outweighed the opinions of the other three pathologists of record. I did not do so for several reasons: First, although Dr. Perper is highly qualified, I find that Dr. Kleinerman is the most qualified pathologist of record, particularly due to his experience with the disease of pneumoconiosis and its pathology. In fact, his curriculum vitae reflects that he was instrumental in the development of the pathology standards for the diagnosis of pneumoconiosis. Insofar as qualifications go, I hold Dr. Mendelow in equal esteem with Dr. Perper. However, I credit his opinion with more weight than Dr. Perper because Dr. Mendelow's opinion was consistent with that of Dr. Kleinerman. I also give less weight to Dr. Perper because he made several findings that no other pathologist made, such as centrilobular emphysema, when all other pathologists, including the prosector, found panlobular or panacinar emphysema. Dr. Perper was also the only physician to have causally related the miner's emphysema to his coal dust exposure. His conclusions regarding the causal relationship of emphysema and coal dust were, in my mind, sufficiently and convincingly rebutted by Dr. Kleinerman's extensive report. I had previously found, and do so now, that the miner's emphysema was directly caused by his heavy smoking history. Even if there was some contribution by his coal dust, it was not sufficient to have caused significant respiratory impairment and certainly cannot be attributed to his death. Dr. Perper was also the only pathologist to have found clinical or medical pneumoconiosis. Dr. Mahmoud noted the presence of nodules but he did not specifically diagnose pneumoconiosis.

I also do not credit Dr. Perper's opinion as to cause of death because he did not fully explain the mechanism or process by which coal workers' pneumoconiosis contributed to death. In fact, he did not specifically state the actual cause of death. Moreover, it does not appear that Dr. Perper was provided with slides of the heart, and consequently, he did not make any diagnoses related thereto, but acknowledged the prosector's diagnoses regarding the heart. Therefore, he did not discuss the effects of the miner's heart disease, even though all three other pathologists implicated heart disease as the primary cause of the miner's death. The reason for this omission is unclear. Dr. Kleinerman and Dr. Mendelow both noted that they were provided with 26 slides, and Dr. Kleinerman provided his findings upon review of the myocardium slides. I find that Dr. Perper did not have enough microscopic data to grasp the full picture of the miner's overall disease processes that were factors in his death. [2]

Normally the prosector would be given a great deal of weight in that he had the opportunity to view the body grossly. Still, a fact finder cannot automatically grant deference to a prosector without a further finding that he had an advantage by having seen gross tissue. In this case, I do not grant the prosector any additional weight over that of the other pathologists, in part because his qualifications are not of record, and it is unclear as to whether he had any clinical records to review. Also, it appears that his findings on microscopic evaluation were consistent with those made by both Drs. Kleinerman and

[2] Although Dr. Mendelow did not provide his findings upon review of the heart slides, he did state that he reviewed all 26 slides; the fact that he diagnosed cardio-respiratory failure and congestive heart failure indicates he was aware of the miner's cardiac problems and its relation to death

Mendelow. There is no evidence that the microscopic findings were not consistent with the gross findings, and I am unwilling to state that the prosector had an advantage over any other physician. Therefore, I hold the prosector on par with the pathologists who only reviewed the microscopic slides.

I had previously held that the cause of death was the miner's coronary artery disease, ASCVD, contributed to by liver cirrhosis. That remains my opinion, as is confirmed by the prosector, Dr. Kleinerman, and Dr. Mendelow. The records from the miner's terminal hospitalization do not implicate any coal dust related disease as a final diagnosis. Emphysema is mentioned but there is no evidence that the treating physician, Dr. Daroski, related this disease to the miner's coal mine employment.

Weighing all of the pathologic evidence together, I continue to find that the opinions of Drs. Kleinerman and Mendelow, supported by the opinion of the prosector, outweigh the opinion of Dr. Perper for the above reasons, i.e., Dr. Perper's lone opinion on several issues; and the greater qualifications of Dr. Kleinerman. Therefore, I continue to find that the miner, even if he had established the presence of pneumoconiosis, did not die due to legal or clinical coal workers' pneumoconiosis, nor did any coal dust related disease substantially contribute to or hasten his death.

*Attorney's Fees*

The award of an attorney's fee under the Act is permitted only in cases in which the claimant is found to be entitled to the receipt of benefits. Because benefits are not awarded in this case, the Act prohibits the charging of any fee to claimant for representation services rendered to him in pursuit of his claim.

CONCLUSION

The claimant has not established the existence of pneumoconiosis arising from coal mine employment nor that the decedent miner died as a result of the disease. Thus, the claimant is not entitled to benefits under the Act and applicable regulations.

**ORDER**

It is ordered that the claim of GRACE POPICH for benefits under the Black Lung Benefits Act is hereby DENIED.

A
RICHARD A. MORGAN
Administrative Law Judge

RAM:HSD:dmr

NOTICE OF APPEAL RIGHTS (Effective Jan. 19, 2001): Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board before the decision becomes "final", i.e., at the expiration of thirty (30) days after "filing" (or receipt by) with the Division of Coal Mine Workers' Compensation, OWCP, ESA, ("DCMWC"), by filing a Notice of Appeal with the Benefits Review Board, ATTN: Clerk of the Board, P.O. Box 37601, Washington, D.C. 20013-7601. A copy of a Notice of Appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, at the Frances Perkins Building, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.